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Mortality in a Region Surrounding an Arsenic Emitting Plant

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The purpose of the investigation is to study whether an increased mortality from certain causes exists in an area around the Rönnskärsverken smelter works in northern Sweden. Founded in 1928, this metallurgical plant processing mainly nonferrous metals has since its initial operations been using ore with a high arsenic content. This has resulted in environmental pollution to air and water of arsenic, as well as other metals and sulfur dioxide.

The causes of death for the population of two parishes in the vicinity of the plant were listed from the National Sedish Register on Death Causes. A reference area in the same part of Sweden with similar degree of urbanization, occupational profile, and age distribution was chosen. The causes of death for the two populations were followed during a period of 14 yr. A significantly higher mortality rate for lung cancer was noted in men in the exposed area. However, this increase was no longer significant when the occupationally exposed at Rönnskärsverken were excluded. The latter showed a highly significant excess mortality due to primary respiratory cancer.

A continuation of this investigation in the form of a cohort study of both the mortality and cancer incidence is currently under way.

Introduction

Exposure to inorganic arsenic, mainly trivalent, has been associated with the development of malignancies, especially in the lung and skin. The development of tumors induced by arsenic has not been achieved as yet in animal experiments (*1*). However, several epidemiological studies have indicated an increased morbidity and mortality in these cancers due to occupational as well as community exposure to arsenic (*2–4*). High levels of arsenic in hair and urine have been recorded in children living near a copper smelter (*5*). Our study was undertaken in order to examine the mortality from different causes in an area surrounding the Rönnskärsverken smelter works in northern Sweden. For several decades this plant has been emitting large amounts of arsenic to air, as well as other metals and sulfur dioxide. The objective was to focus on the community effects, hence excluding the effects on the mortality pattern by the occupationally exposed. This report constitutes the first part of an investigation which will also include a follow-up study and a case-referent study on the same community.

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Methods

Exposure Environment

The Rönnskärsverken smeltery, since its inception in 1928, has been using ores with high arsenic content in the production of lead, copper, zinc, and other metals. This has resulted in emissions where arsenic and sulfur dioxide were the main components. During the period 1930–1960, arsenic was emitted to air in amounts of 1–3 tons/day. Other pollutants of interest were lead, cadmium, mercury, and nickel, although the amounts emitted were much lower than for arsenic. In recent years the emissions of arsenic have decreased, due to the processing of ores with lower content of arsenic, improvements in processing, and installation of dust recovering devices.

The smeltery is located on the tip of a peninsula protruding into the Sea of Bothnia (Fig. 1). The prevailing winds come from the north and south. A previous study regarding the content of heavy metals in top soil and crops indicated the dust fallout as primarily taking place within 10–20 km from the factory (*6*). However, increased levels of mercury in freshwater fish have been reported up to 40 km from the smeltery.

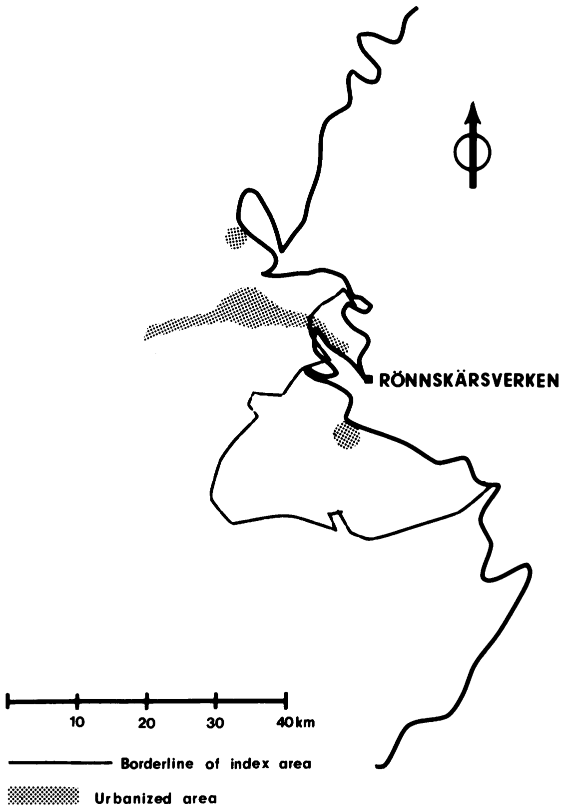

FIGURE 1. The Rönnskärsverken smeltery and the index area.

Selection of Index and Reference Populations

Based on their relation to the prevailing wind directions and the dust fallout area, two parishes near the smeltery were chosen as the exposed area (Fig. 1). The median distance from the factory to the boundary of the exposed area was about 15 km. Because of risk of diluting the effects of pollution, a larger exposed area was not chosen, in spite of the relatively small number of the index population. The total population in the index area in November 1960, was 13,171 persons (*7*).

A reference population with a similar degree of urbanization, occupational profile, fraction of population working, and geographic location was chosen. It consisted of the inhabitants of two parishes on the Sea of Bothnia, 200 km south of the index area. The total reference population in November 1960, was 23,393 persons (*7*).

In Figure 2 are depicted the occupational profiles of the two areas. In both populations the mining and manufacturing industry employed almost 50% of all employed persons. From Figure 3 can be seen that about 40% of the total population in each area was gainfully employed. Also shown is the degree of urbanization for the index and reference populations (67% and 79%, respectively). The age distribution was very similar in the two populations, though the index population was slightly older (Fig. 4).

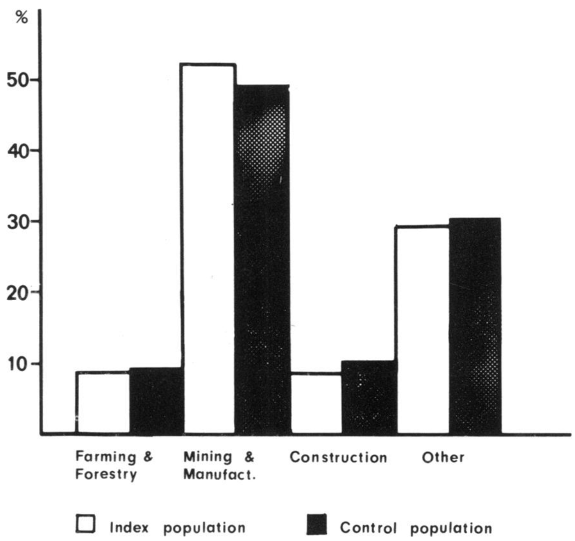

FIGURE 2. Occupational profile in index and control populations. Source: Census in 1960.

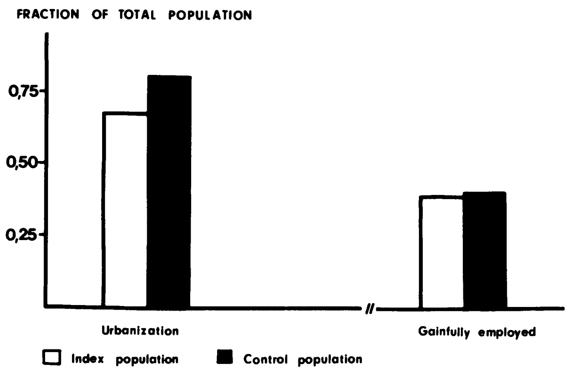

FIGURE 3. Degree of urbanization (inhabitants in municipalities over 200 persons) and the fraction gainfully employed as compared to the total population in the two areas. Source: Census in 1960.

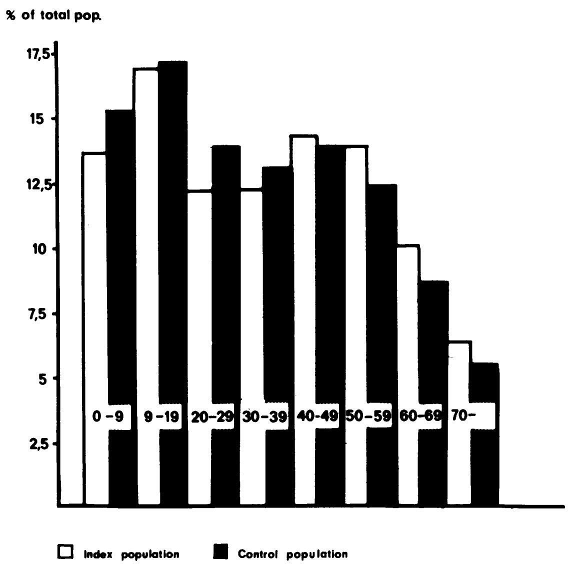

FIGURE 4. Age distribution in index and control populations. Source: Census in 1960, 1965 and 1970.

Source of Material

For the two populations the causes of death were extracted from the National Registry on Causes of Death over a period of fourteen years, January 1, 1961–December 31, 1974. This registry contains information from the death certificate, which in turn contains data from autopsy, hospital diagnoses, etc. The age structure of the investigated populations during the time period at issue was estimated from the National Censuses of 1960, 1965, and 1970.

Methods

The crude mortality rate ratios were calculated for various causes of death according to the 8th revision of the International Classification of Disease (*8*). Employment of the crude rate ratios were feasible for scanning of differences in the pattern of mortality because of the close resemblance in age distribution between the two populations. Tests for significant differences in standardized mortality ratios were performed according to a method suggested by Miettinen (*9*).

Results

No pronounced differences in mortality rates were found among males and females for any causes of death except lung cancer (I.C.D. 162-163) in men (Figs. 5 and 6). Here the exposed population showed a markedly higher crude mortality rate. This could not be explained by differences in age distribution between the two populations as seen below. The excess rates of deaths due to malformations in women as indicated by Figure 6 will be further looked into.

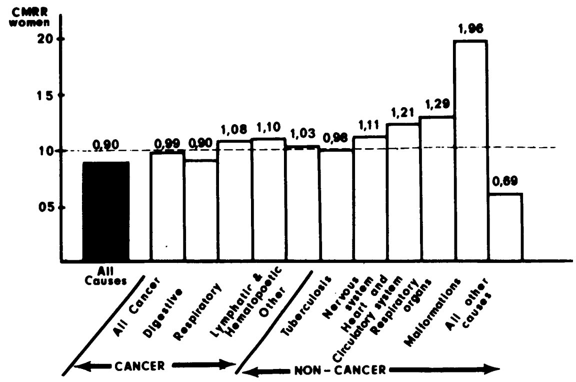

FIGURE 5. Crude mortality rate ratios for selected causes of death in men during the time period 1961–1974.

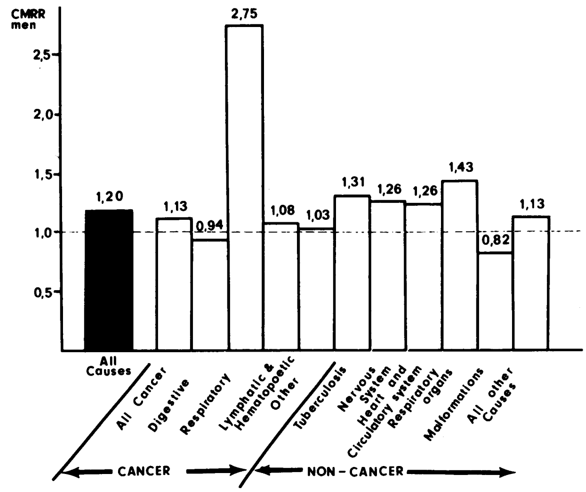

FIGURE 6. Crude mortality rate ratios for selected causes of death in women during the time period 1961–1974.

The standardized mortality ratios (SMR) for primary respiratory cancer in men (I.C.D. 162 and 163.0) were computed by using both rates from the reference area and those from the country as a whole (Table 1). The national rates were based on the annual rates in 1961, 1965, 1970, and 1974 (*10*).

The excess mortality rate in lung cancer in the exposed population was highly significant compared to the mortality in the control population ($p < 0.01$). However, the SMR was not significantly elevated when the national rates were employed.

Table 1. Observed and expected deaths due to primary respiratory cancer in index population with rates from control population and the nation.

		Rates in control area as standard		National rates as standard	
Age	Observed	Expected	SMR	Expected	SMR
40–49	2	1.5		1.0	
50–59	5	1.7		4.1	
60–69	10	4.2		8.6	
70–79	11	3.8		7.8	
Total	28	11.2	250.0[a]	21.5	130.0

[a] $p = 0.0016$ (*9*).

Out of a total of 28 male cases with primary respiratory cancer, 15 had been employed in the Rönnskärsverken smeltery. This industry is the principal employer in the index area and obviously, in focusing on the community effects, the population at risk had to be reduced with the fraction that was or had been working at the smeltery. For this purpose a list was submitted to the company with 113 randomly selected men who had died during the time period at issue at an age over 40. (All lung cancers were found in this age category.) Out of these between 15 and 50% in different age groups had been working at Rönnskärsverken. Using this information a new SMR = 173 could be calculated for the nonoccupationally exposed in this age category. The elevation is not significant; however this SMR represents an underestimation of the rate ratio due to the new nature of the index entity. A large portion of the occupationally exposed with a high risk of dying of lung cancer was excluded from the index population, and no comparable occupational group was excluded from the reference population. The occupationally exposed at Rönnskärsverken showed a substantial excess mortality due to primary respiratory cancer (SMR = 405). Consequently this SMR represents an overestimation of the rate ratio, though due to its magnitude, it should remain highly significant. More valid data regarding the mortality rate ratios for the nonoccupationally exposed will be obtained in the follow-up study currently under way.

Discussion

The observed effects upon mortality for selected causes were similar to that found in previous studies on occupational exposure to arsenic (*3, 4, 11, 12*). The excess lung cancer mortality rate among men were a common feature of all studies. No corresponding increase could be detected among women in this study.

The incidence of lung cancer has been shown to be associated with smoking habits and some social covariables, e.g., degree of urbanization (*13*). The smoking habits in the two populations were not studied as such, but will be studied in future investigations in the area. However it is highly unlikely that they differ substantially in two equally urbanized communities with similar occupational profiles.

The national experience, which is commonly used when computing the SMR, is not adequate in many instances, an example being this study. In our case the influence on the incidence of lung cancer from the more densely populated southern Sweden and the major cities was able to mask the increased mortality in the exposed population. For the purpose of controlling the possible effect of confounding factors the exposed entity should rather be compared to a carefully selected reference population which shares many of the characteristics of the index population except as to the exposure at issue.

The objective of this study was to serve as a pilot study to give an indication of what differences existed and of how to proceed with a more thorough investigation of the patterns of mortality and morbidity in the area around the smeltery. The mortality rates in this study were based on the deaths in the total registered populations 1961–1974 in the two areas and the mean population calculated from the 1960, 1965, and 1970 censuses. Hence there could be a selection bias if the total impact of the smeltery in some way influenced migration to and from the index area. Such a bias should not, however exist in the case of respiratory cancer in view of its rapidly fatal clinical course.

In conclusion, we have found 28 deaths due to primary respiratory cancer in men during the time period 1961–1974 in the index area. This constitutes an excess mortality of between two to three times compared to a similar reference population without the exposure environment of the Rönnskärsverken area. The increase was not significant when the occupationally exposed at Rönnskärsverken were excluded; however in this case the index and reference populations were not comparable as to occupational profile, resulting in an underestimation of the rate ratio as previously noted. A highly significant excess mortality due to primary respiratory cancer among the occupationally exposed at Rönnskärsverken is evident from this study.

The difficulties in establishing causal relationships are well known to epidemiologists. To pinpoint a specific carcinogen is afflicted with great uncertainties in a mixed exposure environment. However, it could well be that it is exactly such a mixture of compounds that is carcinogenic. The inability to induce tumors in animals with arsenic could quite possibly be due to this. There are indications of a cocarcinogenic role of arsenic which could explain some of the effects observed on humans. In the case of skin cancer ultraviolet radiation might serve as the primary carcinogen and regarding lung cancer, components in tobacco smoke are probably the most important determinants. In the area around the smeltery the role of heavy emissions of sulfur dioxide should also be considered.

The concept of a cocarcinogenic activity or arsenic needs further clarification by use of classical toxicological as well as epidemiological methods.

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